Respiratory Function in Pesticide Workers

Eugenija Zuskin, MD, PhD
Jadranka Mustajbegovic, MD, PhD
E. Neil Schachter, MD
Josipa Kern, PhD
Vlasta Deckovic-Vukres, MD, PhD
Ivancica Trosic, PhD
Angelo Chiarelli, BA

Objective: *Pesticide aerosols are frequently toxic irritants associated with respiratory symptoms and lung function impairment.* ***Methods:*** *A cross-sectional study examined the prevalence of acute and chronic respiratory symptoms and lung function abnormalities in 82 workers employed in processing pesticides and in 60 control workers not exposed to irritants and employed in a soft drink bottling plant.* ***Results:*** *The prevalence of almost all chronic respiratory symptoms was greater among pesticide workers than among controls. A logistic regression analysis shows differences between men and women. There was a high prevalence of acute symptoms during the work shift in pesticide workers. The data on ventilatory capacity indicates significant reductions in all tests compared to predicted. Multivariate analysis of lung function showed differences in smoking and work exposure effects in men and women.* ***Conclusion:*** *Our data indicate that duration of work exposure in the pesticide processing industry may be associated with the development of acute and chronic respiratory symptoms and lung function changes. These effects appear to be aggravated by smoking.* (J Occup Environ Med. *2008;50:1299–1305*)

From the Andrija Stampar School of Public Health (Dr Zuskin, Dr Mustajbegovic, Dr Kern), University of Zagreb, Zagreb, Croatia; The Mount Sinai School of Medicine (Dr Schachter, Mr Chiarelli), New York, NY; Croatian Institute for Public Health (Dr Deckovic-Vukres), Zagreb, Croatia; and Institue for Medical Research and Occupational Health (Dr Trosic), Zagreb, Croatia.

Address correspondence to: E. Neil Schachter, MD, The Mount Sinai Hospital, One Gustave L Levy Place, Box 1232, NY 10029-6574; E-mail: neil.schachter@mssm.edu.

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Pesticides are chemical substances that are used for the destruction of environmental organisms deemed detrimental to people.[1] The classical agents include insecticides, fungicides, herbicides, rodenticides, bactericides, miticides, nematocides, and molluscicides. Pesticides can cause intoxication after skin contact, ingestion, or inhalation; moreover, they have the potential to produce both acute and chronic injury to human health.[2] Pesticides, which are toxic by design, are used increasingly in agriculture and in public health programs to control pests and vector-borne diseases. Recent estimates suggest that pesticides account for more than 20,000 fatalities yearly.[3]

Occupational exposure to pesticides occurs in the manufacturing process during storage, during transport, and during application of these products. Exposure occurs among mixers, loaders, and applicators working in fields, greenhouses, parks, and among farmworkers.

There are only a few studies of the respiratory effects of workers involved with pesticides. Results of a study by Hernandez-Valero et al[4] suggest that data obtained from standardized questionnaires may be reasonable indicators of occupational exposure when biomarker data are not available. Respiratory complaints appear common. A case of bronchial asthma due to an organic phosphate insecticide in a laborer employed by a chemical packaging company was described by Weiner.[5] Kossmann et al[6] described that work in the production of pesticides may result in impairment of pulmonary function, development of chronic bronchitis, as well as impaired respiratory muscle function.

In the present study, we investigated respiratory symptoms and lung function in workers employed in a plant processing pesticides to assess the prevalences of respiratory cmplaints among these workers and lung function changes in this setting.

Subjects and Methods

Working Environment

Pesticides processed at the Herbos Plant in Sisak, Croatia are used as insecticides, rodenticides, herbicides, and fungicides primarily for commercial farming and gardening. The pesticides produced in this plant are formulated in liquid, solid, and powdered forms. Synthesis of the pesticides occurs at different locations. The processing in the studied plant consists of three basic steps: mixing, filtration, and drying, followed by homogenization or formulation and packing. Mixing includes the blending of raw materials (these are pesticide chemicals synthesized at other plants eg, organophosphates, carbamates, etc) with addition of alkali (NaOH). Filtration is performed under pressure followed by drying. Drying is accomplished by circulating hot air over the mixture. Homogenization includes thorough mixing of the compouded product.[7]

Subjects

The study included 82 workers (30 female and 52 male) employed in the processing of pesticides. The studied group represents 85% of all workers that were currently employed in this industry. The mean age of the female workers was 44 years (range, 18 to 57 years), their mean height was 161 cm (range, 155 to 171 cm), and the mean duration of exposure in the workplace was 20 years (range, 1 to 41 years). The mean age of the men was 44 years (range, 19 to 59 years), their mean height was 178 cm (range, 160 to 181 cm), and the mean duration of exposure 19 years (range, 2 to 47 years). Among female workers 16 (53.3%) and among male workers 29 (55.8%) were regular smokers. Female workers had smoked on average 7.0 pack years and male workers on average 20.0 pack years. In addition, 25 female workers and 35 male workers employed in a soft drink bottling plant and not exposed to pesticides or other irritants were included in the study as a control group. This population has been previously characterized and acute and chronic symptoms recorded. No lung function was obtained for this control group.[8] We matched our pesticide workers to these control workers based on the best possible match by duration of employment and smoking habit as the exposed pesticide workers (see Table 1).

Our prior studies of men and women in the organic dust industries have demonstrated differences in the response of men and women both in terms of symptom frequency and lung function changes. For this reason, we have focused our analysis on possible gender differences in this industry.

Respiratory Symptoms

Chronic respiratory symptoms were recorded by physicians using the British Medical Research Council questionnaire on respiratory symptoms[9] with additional questions on occupational asthma.[10–12] In pesticide as well as in control workers, a detailed occupational history and questions about their smoking habit were recorded. The following definitions were used:

Chronic cough or phlegm: cough and/or phlegm for a minimum of 3 months a year.

Chronic bronchitis: cough and phlegm for a minimum of 3 months a year and for not less than 2 successive years.

Dyspnea grades: 3, shortness of breath when walking with other people at an ordinary pace on level ground; and 4, shortness of breath when walking at one's own pace on level ground.

Occupational asthma: industrial physician diagnosed asthma in the workplace setting.

TABLE 1
Chronic Respiratory Symptoms in Pesticide and Control Workers

Sex	Group	Age (yrs)	Employment (yrs)	Smoking (Pack Year)	Chronic Cough	Chronic Phlegm	Chronic Bronchitis	Occupational Asthma	Dyspnea Grades 3 and 4	Sinusitis	Nasal Catarrh	Hoarseness
Female	Pesticide *N* = 30	44 ± 7	20 ± 7	17.6 ± 8	11 (36.7%)	7 (23.3%)	7 (23.3%)	1 (3.3%)	8 (26.7%)	8 (26.7%)	12 (40.0%)	15 (50.0%)
	P-Value of difference	NS	NS	NS	<0.05	NS	NS	NS	<0.05	<0.05	<0.01	<0.01
	Control *N* = 25	43 ± 6	20 ± 6	16.5 ± 7	3 (12.0%)	2 (8.0%)	2 (8.0%)	0 (0%)	1 (4.0%)	1 (4.0%)	0 (0%)	0 (0%)
Male	Pesticide *N* = 52	44 ± 11	19 ± 11	24.6 ± 18	(1732.7%)	16 (30.8%)	16 (30.8%)	1 (1.9%)	3 (5.8%)	24 (46.2%)	21 (40.4%)	13 (25.0%)
	P-Value of difference	NS	NS	NS	<0.05	<0.05	<0.05	NS	NS	<0.01	<0.01	<0.01
	Control *N* = 35	40 ± 10	17 ± 9	22.3 ± 16	5 (14.3%)	4 (11.4%)	4 (11.4%)	0 (0%)	1 (2.9%)	2 (5.7%)	1 (2.9%)	0 (0%)

NS, difference statistically not significant ($P > 0.05$).

Acute symptoms that developed during the work shift were also recorded using a questionnaire in all pesticide and control workers by a physician during the period of the study. Acute symptoms included dry cough, shortness of breath, irritation of the throat, dryness of the throat, irritation of the nose, dryness of the nose, eye irritation, and headache.

Ventilatory Capacity

Ventilatory capacity measurements were performed in pesticide workers by recording the maximum expiratory flow-volume (MEFV) curves using a pneumotach spirometer (Pneumoscreen, Jaeger, Germany). The forced vital capacity (FVC), the forced expiratory volume in the first second (FEV1), and the maximum forced expiratory flow (FEF) at 50% and the last 25% of the FVC (FEF50, FEF25) were measured on the MEFV curves. Measurements were performed during the morning work shift. The spirometer was calibrated on a daily basis. Lung function testing was performed according to the recommendations of Quanjer et al.[13] At least three MEFV curves were recorded for each subject and the best value of three technically satisfactory MEFV curves was used as the result of the test (this was the curve with the greatest FVC and FEV1). The measured ventilatory capacity was compared with the predicted normal values of Quanjer et al[14] which are based on sex, age, and height. Lung function testing was not performed in the group of female and male control workers as these have been previously compared to predicted and found to be normal.[15]

Statistical Analysis

The differences in the prevalence of acute and chonic respiratory symptoms recorded in pesticide and control workers, as well as between smokers and nonsmokers were tested by the χ^2 test (or when appropriate, Fisher exact test). Odds ratios were calculated by using a logistic regression analysis for each respiratory symptom (age, length of exposure, and smoking were the predictors).[16] The results of ventilatory capacity measurements were analyzed by the paired *t* test when comparing baseline with predicted values. Ventilatory capacity data were also analyzed by applying a multiple regression analysis with age, exposure, and smoking as predictors and % predicted of FVC, FEV1, FEF50, and FEF25 as dependant variables.[17] A level of $P < 0.05$ was considered statistically significant. Analyses were stratified by gender.

Results

Respiratory Symptoms

The prevalence of chronic respiratory symptoms in pesticide and in control workers is shown in Table 1. For most of the chronic respiratory symptoms, the prevalences were significantly higher in the pesticide workers than in the controls ($P < 0.05$ or $P < 0.01$). Occupational asthma was diagnosed in one female pesticide worker (3.3%), in one male pesticide worker (1.9%), and in none of the control workers (not significant).

Table 2 shows the results of a logistic regression analysis for individual chronic symptoms in female and male pesticide workers. Significant odds ratios were seen as a result of work exposure for chronic cough, dyspnea, and nasal catarrh for women, and for dyspnea and nasal catarrh in men.

The prevalences of acute symptoms during the work shift in pesticide workers are presented in Table 3. Large numbers of workers com-

TABLE 2
Logistic Regression Analysis for Chronic Respiratory Symptoms by Gender Using Age, Exposure, and Smoking as Predictor Variables

	Odds Ratio					
	Female (*N* = 30)			Male (*N* = 52)		
Symptoms	Age	Exposure	Smoking	Age	Exposure	Smoking
Chronic cough	0.89 (0.68–1.10)	1.29* (1.15–15.84)	0.95 (0.86–1.03)	1.07 (0.94–1.23)	0.98 (0.86–1.11)	1.06 (1.02–1.11)
Chronic phlegm	0.98 (0.78–1.22)	1.04 (0.81–1.36)	1.02 (0.94–1.11)	1.04 (0.92–1.16)	1.03 (0.93–1.15)	1.02 (0.99–1.06)
Chronic bronchitis	0.98 (0.78–1.22)	1.04 (0.81–1.36)	1.02 (0.94–1.11)	1.04 (0.92–1.16)	1.03 (0.93–1.15)	1.02 (0.99–1.06)
Dyspnea grades 3 and 4	0.78 (0.61–1.10)	1.11* (1.06–1.97)	1.21* (1.09–2.98)	1.12 (0.90–3.15)	2.35* (1.50–4.10)	0.70 (0.10–1.15)
Sinusitis	1.27* (1.02–11.66)	0.75 (0.53–0.98)	0.98 (0.89–1.07)	0.10 (0.90–1.10)	1.00 (0.91–1.11)	0.99 (0.95–1.02)
Nasal catarrh	1.91* (1.10–3.10)	2.08* (1.12–3.40)	1.81* (1.10–2.95)	0.95* (0.15–1.10)	2.15* (1.15–4.10)	1.15* (1.01–3.15)
Hoarseness	1.09 (0.90–1.34)	1.03 (0.83–1.29)	0.99 (0.89–1.07)	1.01 (0.90–1.13)	1.01 (0.92–1.14)	1.02 (0.98–1.05)

*Statistically significant ($P < 0.05$); 95% confidence interval (CI) in parentheses.

TABLE 3
Acute Symptoms During Work Shift in Pesticide Workers

Sex	Cough	Wheezing	Chest Tightness	Dyspnea	Throat Irritation	Throat Dryness	Eye Irritation	Nose Secretion	Nose Dryness	Nose Bleeding	Headache
Female	23 (76.7%)	13 (43.3%)	14 (46.7%)	22 (73.3%)	23 (76.7%)	22 (73.3%)	25 (83.3%)	10 (33.3%)	17 (56.7%)	7 (23.3%)	23 (76.7%)
(N = 30)	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.05	<0.01	<0.01
Male (N = 52)	15 (28.9%)	4 (7.7%)	9 (17.3%)	22 (42.3%)	18 (34.6%)	19 (36.5%)	23 (44.2%)	3 (5.8%)	18 (34.6%)	2 (3.9%)	12 (23.1%)

NS, difference statistically not significant ($P > 0.05$).

plained of these acute symptoms and differences between men and women are noted. Table 4 shows the results of a logistic regression analysis for individual acute symptoms in female and male pesticide workers. Significant odds ratios were seen as a result of work exposure for dyspnea, eye irritation, nasal secretion, and nasal dryness for women and for dyspnea, throat irritation, eye irritation, nasal dryness, and epistaxis in men.

Ventilatory Capacity

Table 5 presents ventilatory capacity data for female and male pesticide workers as measured values and predicted[14] and the difference between measured and predicted. The measured FVC, FEF50, and FEF25 were significantly lower when compared to predicted in both men and women pesticide workers.

The findings of our multiple regression analysis with duration of work exposure and amount smoked as predictors and lung function parameters as outcome variables are shown for female pesticide workers in Table 6, and for male pesticide workers in Table 7. Among women, smoking approached significance as a cause of lung function abnormality for FEF50 and FEF25, whereas duration of work exposure was significant for FEF25. In men, duration of work exposure was strongly linked to abnormalities in all lung function parameters whereas smoking was significant for FEF25 and FEF50.

Analysis of the individual measured lung function data in pesticide workers showed that values lower than 70% of predicted for FVC and FEV1 were found in 3.3% and 16.0% of the studied workers, respectively and for FEF50 and FEF25 in 10.0% and 18.0% of the workers, respectively. Similar frequencies of lung function tests less than 70% of predicted were found in female and male pesticide workers.

Discussion

Our data indicate that occupational exposure to pesticides in the processing of these materials may be responsible for the development of acute and chronic respiratory symptoms accompanied by impairment of ventilatory capacity.

Many reports over the past decade document possible differences between the development of chronic lung disease in men and women.[18,19] The rationale for such differences span the spectrum of intrinsic (eg, hormonal) causes to environmental (eg, exposure) differences. Some of these questions may be fruitfully addressed in the workplace.

There are some striking similarities and differences between men and women in terms of symptoms and lung function as outlined in Tables 2, 4, 6, and 7.

In terms of chronic symptoms both men and women appear to display similar increases in dyspnea and nasal catarrh associated with length of exposure. Acute symptoms which are a response to the question: do you usually experience X symptom during working hours indicates that there is a definite association between exposure length and upper airway symptoms relating to the eye, nose, and throat in both men and women. This may suggest the presence of a water soluble, irritant agent in the work environment which would be more likely to affect the moist mucosa of the upper airway.

Finally, there is a distinct difference between men and women as to lung function loss and its association with exposure. In this study, men were more likely to experience widespread lung function loss associated with exposure than women workers. This may point to either an intrinsic gender difference rendering men more susceptible, or some difference in workplace exposures based on sex. Our current investigation does not permit us to better define these factors better.

With the increased use of pesticides in both agricultural and urban environments, interest has focused on the health effects of these agents. Individuals using these products have been studied. A history of respiratory problems, particularly bronchitis, in farm-

TABLE 4
Logistic Regression Analysis for Acute Respiratory Symptoms by Gender Using Age, Exposure, and Smoking as Predictor Variables

	Odds Ratio					
	Female (N = 30)			Male (N = 52)		
Symptoms	Age	Exposure	Smoking	Age	Exposure	Smoking
Acute cough	1.08 (0.85–1.41)	1.09 (0.81–1.50)	1.12* (1.02–5.40)	0.99 (0.88–1.09)	1.00 (0.903–1.110)	1.09 (0.97–1.04)
Wheezing	0.99 (0.82–1.20)	1.08 (0.87–1.37)	0.98 (0.92–1.06)	0.93 (0.73–1.10)	1.04 (0.89–1.28)	1.06 (0.99–1.44)
Chest tightness	0.99 (0.83–1.19)	1.04 (0.84–1.30)	0.99 (0.43–1.10)	0.94 (0.78–1.07)	1.06 (0.94–1.29)	1.02 (0.98–1.07)
Dyspnea	0.78 (0.61–1.10)	1.11* (1.06–1.97)	1.21* (1.09–2.98)	0.97 (0.67–1.91)	2.78* (1.90–4.15)	1.70* (1.15–3.70)
Throat irritation	0.15 (0.10–1.95)	0.25 (0.10–0.90)	0.57 (0.15–1.10)	0.85 (0.32–1.10)	1.36* (1.10–3.50)	1.48* (1.09–3.10)
Eye irritation	0.10* (0.05–1.90)	1.10* (1.05–2.96)	0.80 (0.10–0.99)	0.90 (0.10–1.12)	1.45* (1.12–3.10)	0.76 (0.57–0.90)
Nasal secretion	0.50 (0.10–1.50)	1.20* (1.09–3.10)	0.51 (0.10–1.10)	1.10 (0.90–1.15)	0.90 (0.70–1.10)	1.19 (0.70–1.11)
Nasal dryness	0.75 (0.11–1.90)	1.15* (1.05–2.91)	0.90 (0.51–1.10)	0.90 (0.10–1.12)	1.19* (1.10–3.15)	0.16 (0.10–1.90)
Nasal bleeding	1.05 (0.84–1.32)	1.02 (0.80–1.31)	1.01 (0.95–1.13)	0.801 (0.33–1.39)	1.40* (1.21–13.56)	0.926 (0.69–1.03)

*Statistically significant ($P < 0.05$); 95% confidence interval (CI) in brackets.

ing and nonfarming households related to the use of pesticides has been described by Masley et al.[20] The effect of insecticide aerosols on lung function, airway responsiveness, and symptoms in asthmatic subjects was described by Salome et al.[21] The authors concluded that insecticide aerosols trigger symptoms and decrease lung function in some asthmatics. These aerosols may also increase airway hyperresponsiveness. Senthilselvan et al[22] reported an association of asthma with use of pesticides by farmers. Wilkins et al[23] described frequent cough related to exposure to pesticides among Ohio grain farmers. Our study by contrast focusing on workers who prepare pesticides indicates that they are also at risk.

Symptoms, such as irritation of the eyes, dyspnea, cough, and headache due to exposure to pesticides were described by Rico Mendez et al.[24] Similarly, in our study of agricultural farmers, we found respiratory impairment including acute symptoms from upper respiratory system and eye irritation and headache which may be partly related to the use of pesticides.[15] Martin et al[25] studied pesticide use and pesticide-related symptoms among black and white agricultural farmers and reported increased symptoms such as headaches, dizziness, skin irritation, chest discomfort, and feeling nervous or depressed.

Smoking indoors may confound work environment effects. This did not appear to be a major issue in this study for chronic respiratory symptoms as there was little difference between smoking and nonsmoking workers (Table 2). The possibility of self-selection in small group must be considered; however, in view of the high level of participation in this study this would seem unlikely. The low level of asthma in all groups (pesticide workers and controls) on the other hand may indicate a healthy worker effect.

Occupational exposure to pesticides occurs mainly as the result of inadequate personal protective equipment, and of training in the safe management of chemicals.[26] Workers tend not to use protective measures while handling pesticides.[27] Handwashing and showering are important ways to reduce pesticide exposure. In addition, workers should not bring their work clothes home and should wash these clothes separately to avoid contamination.[28]

We have shown that exposure to toxins in the pesticide processing industry can result in measurable changes in symptoms and lung function. Preventive measures should include worker safety and education programs, medical surveillance for early identification of affected workers and screening for sensitive individuals.

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TABLE 5
Ventilatory Capacity in Pesticide Workers

			FVC			FEV1			FEF50			FEF25		
Sex	Mean Age (yrs)	Mean Height (cm)	Measured (L)	Predicted (%)	Difference Measured–Predicted (*P**)	Measured (L)	Predicted (%)	Difference Measured–Predicted (*P**)	Measured (L/s)	Predicted (%)	Difference Measured–Predicted (*P**)	Measured (L/s)	Predicted (%)	Difference Measured–Predicted (*P**)
Female *N* = 30	44 ± 7	161 ± 6	3.2 ± 0.5	93.9 ± 11.0	<0.01	2.7 ± 0.4	99.8 ± 12.6	NS	3.6 ± 0.8	87.0 ± 19.4	<0.01	1.6 ± 0.5	75.8 ± 20.9	<0.01
Male *N* = 52	44 ± 11	178 ± 7	4.7 ± 1.0	95.4 ± 12.8	<0.05	3.9 ± 0.8	100.5 ± 13.2	NS	4.6 ± 1.5	88.3 ± 26.7	<0.01	1.9 ± 0.8	75.9 ± 26.3	<0.01

*Differences are between actual measured and predicted values. Percent predicted is included to benchmark the actual values.
The measured data are presented as mean ± SD.
NS, difference statistically not significant ($P > 0.05$).

TABLE 6
Regression Analysis of Ventilatory Capacity Tests in 30 Female Pesticide Workers

Test	Variable	*df*	Parameter Estimate (β)	Standard Error (SE)	T for HO: Parameter = 0	Prob >(T) (*P*)
FVC	Exposure*	1	−0.01	0.02	−0.74	0.4619
	Smoking†	1	0.01	0.01	0.61	0.5500
FEV1	Exposure	1	−0.01	0.01	−0.98	0.3363
	Smoking	1	−0.00	0.01	−0.55	0.5872
FEF50	Exposure	1	−0.00	0.03	−0.18	0.8508
	Smoking		−0.03	0.01	−1.99	0.0568
FEF25	Exposure	1	−0.03	0.01	−2.60	0.0150
	Smoking	1	−0.01	0.01	−1.95	0.0623

*Exposure = length of time in the industry.
†Smoking = pack year.
T—*t*-statistic for the null hypothesis.
HO—that implies the parameter to be 0.

TABLE 7
Regression Analysis of Ventilatory Capacity Tests in 52 Male Pesticide Workers

Test	Variable	*df*	Parameter Estimate (β)	Standard Error (SE)	T for HO: Parameter = 0	Prob >(T) (*P*)
FVC	Exposure	1	−0.05	0.01	−4.065	0.0001
	Smoking	1	0.00	0.01	0.591	0.5564
FEV1	Exposure	1	−0.04	0.01	−4.750	0.0001
	Smoking	1	−0.00	0.01	−0.405	0.6867
FEF50	Exposure	1	−0.05	0.02	−2.954	0.0041
	Smoking	1	−0.02	0.01	−2.026	0.0461
FEF25	Exposure	1	−0.03	0.01	−4.436	0.0001
	Smoking	1	−0.01	0.00	−2.163	0.0336

T—*t*-statistic for the null hypothesis.
HO—that implies the parameter to be 0.

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